(2)

Report No. NADC-91042-60

AD-A239 561

THE G-LOC SYNDROME

James E. Whinnery, M.D., Ph.D.
Air Vehicle and Crew Systems Technology Department (Code 602C)
NAVAL AIR DEVELOPMENT CENTER
Warminster, PA 18974-5000

31 OCTOBER 1990

FINAL REPORT

Approved for Public Release; Distribution is Unlimited

Prepared for
Air Vehicle and Crew Systems Technology Department (Code 602C)
NAVAL AIR DEVELOPMENT CENTER
Warminster, PA 18974-5000

NOTICES

REPORT NUMBERING SYSTEM — The numbering of technical project reports issued by the Naval Air Development Center is arranged for specific identification purposes. Each number consists of the Center acronym, the calendar year in which the number was assigned, the sequence number of the report within the specific calendar year, and the official 2-digit correspondence code of the Command Officer or the Functional Department responsible for the report. For example: Report No. NADC-88020-60 indicates the twentieth Center report for the year 1988 and prepared by the Air Vehicle and Crew Systems Technology Department. The numerical codes are as follows:

CODE	OFFICE OR DEPARTMENT
00	Commander, Naval Air Development Center
01	Technical Director, Naval Air Development Center
05	Computer Department
10	AntiSubmarine Warfare Systems Department
20	Tactical Air Systems Department
30	Warfare Systems Analysis Department
40	Communication Navigation Technology Department
50	Mission Avionics Technology Department
60	Air Vehicle & Crew Systems Technology Department
70	Systems & Software Technology Department
80	Engineering Support Group
90	Test & Evaluation Group

PRODUCT ENDORSEMENT — The discussion or instructions concerning commercial products herein do not constitute an endorsement by the Government nor do they convey or imply the license or right to use such products.

Reviewed By: James E. Whinnery Ph.D., M.D. Date: 30 May 1991
Branch Head

Reviewed By: L H Israel, Ph.D Date: 3 June 91
Division Head

Reviewed By: W J Smith Date: 6/4/91
Director/Deputy Director

UNCLASSIFIED
SECURITY CLASSIFICATION OF THIS PAGE

REPORT DOCUMENTATION PAGE

Form Approved
OMB No 0704-0188

1a REPORT SECURITY CLASSIFICATION Unclassified	1b RESTRICTIVE MARKINGS
2a SECURITY CLASSIFICATION AUTHORITY	3 DISTRIBUTION / AVAILABILITY OF REPORT Approved for Public Release; Distribution is Unlimited
2b DECLASSIFICATION / DOWNGRADING SCHEDULE	
4 PERFORMING ORGANIZATION REPORT NUMBER(S) NADC-91042-60	5 MONITORING ORGANIZATION REPORT NUMBER(S)
6a NAME OF PERFORMING ORGANIZATION Air Vehicle and Crew Systems Technology Department 6b OFFICE SYMBOL (If applicable) 602C	7a NAME OF MONITORING ORGANIZATION
6c ADDRESS (City, State, and ZIP Code) NAVAL AIR DEVELOPMENT CENTER Warminster, PA 18974-5000	7b ADDRESS (City, State, and ZIP Code)
8a NAME OF FUNDING / SPONSORING ORGANIZATION Air Vehicle and Crew Systems Technology Dept. 8b OFFICE SYMBOL (If applicable) 602C	9 PROCUREMENT INSTRUMENT IDENTIFICATION NUMBER
8c ADDRESS (City, State, and ZIP Code) NAVAL AIR DEVELOPMENT CENTER Warminster, PA 18974-5000	10 SOURCE OF FUNDING NUMBERS PROGRAM ELEMENT NO / PROJECT NO / TASK NO / WORK UNIT ACCESSION NO

11 TITLE (Include Security Classification)
The G-LOC Syndrome

12 PERSONAL AUTHOR(S)
James E. Whinnery, M.D., Ph.D.

13a TYPE OF REPORT	13b TIME COVERED	14 DATE OF REPORT (Year, Month Day)	15 PAGE COUNT
Final	FROM _____ TO _____	1990 October 31	

16 SUPPLEMENTARY NOTATION

17 COSATI CODES			18 SUBJECT TERMS (Continue on reverse if necessary and identify by block number)
FIELD	GROUP	SUB-GROUP	Unconsciousness, Fighter Aviation, Acceleration (+G_z) Centrifuge, Neurophysiology, Ischemia
01	03	03	

19 ABSTRACT (Continue on reverse if necessary and identify by block number)

Acceleration (+G_z)-induced loss of consciousness (G-LOC) is but one isolated symptom that results when central nervous system (CNS) function is altered by G-stress. Other symptoms and changes also result when reduction of oxygenated blood flow to the CNS occurs. The complex of symptoms and changes that result from G-stress occur in close temporal relationship and therefore form a G-LOC syndrome. Recognition of the G-LOC syndrome serves to include the associated symptoms as part of the normal response to CNS ischemia/hypoxia. This recognition is important in reducing the potential for unnecessary aeromedical evaluation or disqualification of normal aircrew who have an uncomplicated G-LOC episode. It also serves to ensure that abnormal responses can be more clearly identified. Recognition of the G-LOC syndrome also serves to enhance the understanding that G-LOC is a primary neurologic disturbance induced by G-stress. We propose to clarify the existing situation by recognizing the G-LOC syndrome as including all of the +G_z- induced CNS symptoms, either alone or in combination, as a normal response to +G_z-stress in normal human beings.

20 DISTRIBUTION AVAILABILITY OF ABSTRACT	21 ABSTRACT SECURITY CLASSIFICATION
☒ UNCLASSIFIED UNLIMITED ☐ SAME AS RPT ☐ DTIC USERS	Unclassified
22a NAME OF RESPONSIBLE INDIVIDUAL James E. Whinnery, M.D., Ph.D.	22b TELEPHONE (Include Area Code) 215-441-1967 22c OFFICE SYMBOL 602C

FIGURES

Acceleration ($+G_z$) induced loss of consciousness (G-LOC) results when an individual is exposed to a level of $+G_z$-stress for a period of time that exceeds his body's neurologic tolerance to such stress. $+G_z$-stress results in a downward displacement of blood away from the head. This may result in sufficient ischemia to alter normal conscious function within the central nervous system (CNS) if the stress persists for a sufficiently long period. Alterations in pulmonary ventilation-perfusion may also result in hypoxic insult to the CNS. It is for these reasons that G-LOC may result from either ischemia or hypoxia or a combination of both ischemia/hypoxia, depending on the characteristics of the $+G_z$ exposure profile.

Burton previously defined G-LOC as the following (1): "a state of altered perception wherein (one's) awareness of reality is absent as a result of sudden, critical reduction of cerebral blood circulation caused by increased G force." Based on the characteristics of symptoms associated with G-LOC, modification of that definition was recommended to indicate that the $+G_z$-induced ischemic/hypoxic insult is not limited to the cerebral cortex (2). The definition of G-LOC therefore becomes "a state of altered perception wherein (one's) awareness of reality is absent as a result of sudden, critical reduction of central nervous system circulation caused by increased G force."

It is important to carefully understand that G-LOC refers only to the loss of conscious function. This is the highest of neurologic functions. Consciousness is a separate psychophysiologic state. Although intricately intertwined to other $+G_z$-induced ischemic/hypoxic symptoms, consciousness does not per se include them. It is considered a state in which a person is not aware of any sensory impressions and has no subjective experiences. This is an extremely difficult condition to accurately measure even though it can be overtly evident when it is observed to exist in an individual. One person's knowledge of another's state of consciousness is frequently only an inference based on observations of motor activity. Based on the above description of unconsciousness, it is essentially impossible to subjectively or objectively determine exactly when someone else becomes unconsciousness or recovers consciousness. Perhaps individuals like fighter aircrew, whose very profession depends on maintaining consciousness, are an ideal population in which to evaluate loss of consciousness phenomena. They

are extremely motivated to maintain consciousness and, should it be lost, they are exceptionally eager to demonstrate the moment it returns. If we are to eventually be able to assess whether someone has lost consciousness, it is important to describe in agonizing detail and as completely as possible all of the observable characteristics associated with an unconsciousness episode.

When one endeavors to understand loss of consciousness it becomes evident that it is so intimately associated with other CNS functions that exclusive isolation of consciousness is difficult. Consciousness results from fully integrated and functional circuits within the CNS. Any breakdown or alteration of this integration may affect consciousness. Other functions may be concurrently altered or altered in close temporal association with G-LOC. The ischemic/hypoxic insult to the CNS that produces G-LOC therefore frequently produces a complex of symptoms and characteristics and not just isolated loss of consciousness. It is for this reason that the phenomena observed in association with $+G_z$-induced ischemia/hypoxia of the CNS would be more appropriately termed the G-LOC syndrome. Syndrome comes from the Greek *syndrome* meaning a running together, which is exactly what the symptoms and characteristics of $+G_z$-induced ischemia/hypoxia certainly do. This terminology successfully isolates unconsciousness as a discreet part of the entire syndrome. The G-LOC syndrome would include not only the loss of consciousness but also the loss of vision, loss of muscle control, convulsive activity, dream phenomenon, altered memory, and other symptoms which may occur in close association. As such, the G-LOC syndrome constitutes all the symptoms and characteristics resulting from the induction of and recovery from $+G_z$-induced ischemic/hypoxic insult to the nervous system (Figure 1). By definition, the G-LOC syndrome is the spectrum of neuro- and psycho-physiological changes and symptoms that result from G-induced alterations in the supply of oxygenated blood to the central nervous system. Although Figure 1 lists only the currently known observable symptoms, the G-LOC syndrome includes the objectively measured changes such as those observed using electroencephalography. It is important to realize that the known symptoms and changes associated with the G-LOC syndrome are produced from a relatively narrow range of $+G_z$-induced ischemic/hypoxic exposures. Additional symptoms and changes are possible as other exposure

envelopes are investigated and newer experimental measurement techniques are developed. It would perhaps be more appropriate to call the spectrum of changes and symptoms something other than the G-LOC syndrome since indeed G-LOC need not be a part of what happens on every exposure. The alternatives might be the G-induced neurologic alteration syndrome or the G-induced hypoxia/ischemia syndrome (or others). However, G-LOC is a well established term, is a fighter aviation critical event and, therefore is not unreasonable for reason of simplicity.

An additional consideration is the terminology involving G-LOC itself. Strictly speaking $+G_z$-induced loss of consciousness refers only to the loss of consciousness and not necessarily the subsequent unconscious period. The loss of consciousness initiates the unconsciousness period. It is a point or spanse of time that is only the initiating event associated with $+G_z$-induced unconsciousness. $+G_z$-induced unconsciousness is perhaps more appropriate terminology for the unconsciousness period. The previous definitions of G-LOC would therefore be more appropriately applied to +Gz-induced unconsciousness. The strict definition of G-LOC in a kinetic sense would be: "The point or spanse of time wherein $+G_z$-induced ischemia/hypoxia results in a transition from consciousness to unconsciousness."

With respect to the definition of G-LOC (or what is more appropriately termed $+G_z$-induced unconsciousness), the following definition is suggested as a modification of that previously agreed upon: a state of altered perception wherein (one's) awareness of reality is absent following a sudden, critical, G-induced reduction of oxygenated central nervous system blood flow causing interruption of normal integrated neurologic function. It is important to understand that G-LOC, $+G_z$-induced unconsciousness, and the G-LOC syndrome are neurologic phenomenon caused by alteration of neurologic function. This is preceded by alteration of the cardiovascular system but the primary failure is neurologic. The cardiovascular system does not generally fail in G-LOC. It is working normally. Although methods and techniques to support the cardiovascular system have been important for reducing the potential for G-LOC and the G-LOC syndrome, they cannot absolutely prevent them. A neurologic tolerance enhancement technique on the other hand, although ambitious, could prevent these alterations. The

above definition of G-LOC is an attempt to enhance the notion that interruption of integrated neurologic function is the key factor in this phenomenon.

The G-LOC syndrome associated with a rapid-onset, sustained high $+G_z$ profile is shown in Figure 2. The complexity of the entire syndrome is evident. The importance of such detailed analysis and the requirement for careful definition of all terminology cannot be overstated if G-LOC is to yield to an ultimate understanding (3,4). Such analysis is tedious and requires considerable consistency. This complexity can be illustrated by examining what we have defined as the absolute incapacitation period as illustrated in Figure 3 (5). This was called absolute incapacitation and loosely equated with the period of unconsciousness. Refraining from calling this period unconsciousness was purposeful. Absolute incapacitation is an operational measurement subjectively determined. It can be measured by observing when an individual becomes abruptly unresponsive and coincidentally loses postural tone. Exactly when the individual actually loses consciousness may be somewhat earlier than what can be subjectively observed or even objectively measured. Just how accurately one requires to determine the onset of unconsciousness depends on the acceptability of such subjective measurements. For operational fighter aviation medical purposes, this method is acceptable since it is probably within at least 1s of the onset of unconsciousness. The same problem exists relative to exact determination of the recovery of consciousness. In addition, return of consciousness is very dependent on the definitions employed. Our subjective measure of the moment of return of consciousness is determined by observing the subject to be capable of being responsive. This can usually be done within a 1s to 2s accuracy. However, if unconsciousness is defined as a state in which a person is not aware of any sensory impressions and has no subjective experiences, then we are actually over estimating the duration unconsciousness. When the subject experiences a dream he is technically conscious according to such a definition. The "absolutely incapacitated" individual has a subjective experience when he dreams. As such, he would be "conscious" at this time. The error in such instances depends on knowing when and how long the dream period is. This is not currently possible. It also depends on how we define unconsciousness. The entire G-LOC syndrome is of very short duration, if we ignore any persistent

psychophysiologic effects. Such brevity complicates the ability to make all the measurements of all the events in so short a time period. However, if we do not endeavor to at least begin the crude description now, we could never hope to design the experiments and clever devices to accurately define the complete G-LOC syndrome.

One of the major considerations for establishing the terminology "G-LOC syndrome" is to ultimately protect fighter aircrew by reducing the potential for subjecting them to unnecessary aeromedical evaluation or flight duty restriction. The symptoms and characteristics of the G-LOC syndrome are a "mini-simulation" of a wide variety of CNS diseases and abnormalities. They are not, however, indicative of any abnormality and frequently represent the normal functioning of CNS centers released (disinhibited) from their usual control. Regional ischemic differences within the CNS are established by $+G_z$-stress and dictate the nature and extent of the G-LOC syndrome which will be manifest in a healthy fighter pilot. By recognizing that such characteristics as unconsciousness, convulsive activity, and electroencephalographic alterations are all part of the G-LOC syndrome, it recognizes them as being normal responses in this environment and not reason for disqualification or further aeromedical evaluation. It is probable that additional G-LOC syndrome symptoms will be discovered as different $+G_z$-profiles are developed and induce unique configurations of regional CNS ischemic differential. A detailed understanding of the characteristics and mechanism of $+G_z$-induced ischemia/hypoxia in completely normal humans is key for supporting fighter aircrew. Such support is the absolute core of our duty as fighter aviation medicine subspecialists.

REFERENCES

1. Burton RR. G-induced loss of consciousness: definition, history, current status. Aviat. Space Environ. Med. 59:2-5; 1988.

2. Whinnery JE. Defining risk in aerospace medical unconsciousness research. Aviat. Space Environ. Med. 60: 688-694; 1989.

3. Whinnery JE. Methods for describing and quantifying $+G_z$-induced loss of consciousness. Aviat. Space Environ. Med. 60:798-802; 1989.

4. Whinnery JE, Whinnery AM. Acceleration-induced loss of consciousness. Arch. Neurol. 47:764-776; 1990.

5. Whinnery JE, Burton RR, Boll PA, Eddy DR. Characteristics of the resulting incapacitation following unexpected $+G_z$-induced loss of consciousness. Aviat. Space Environ. Med. 58:631-636; 1987.

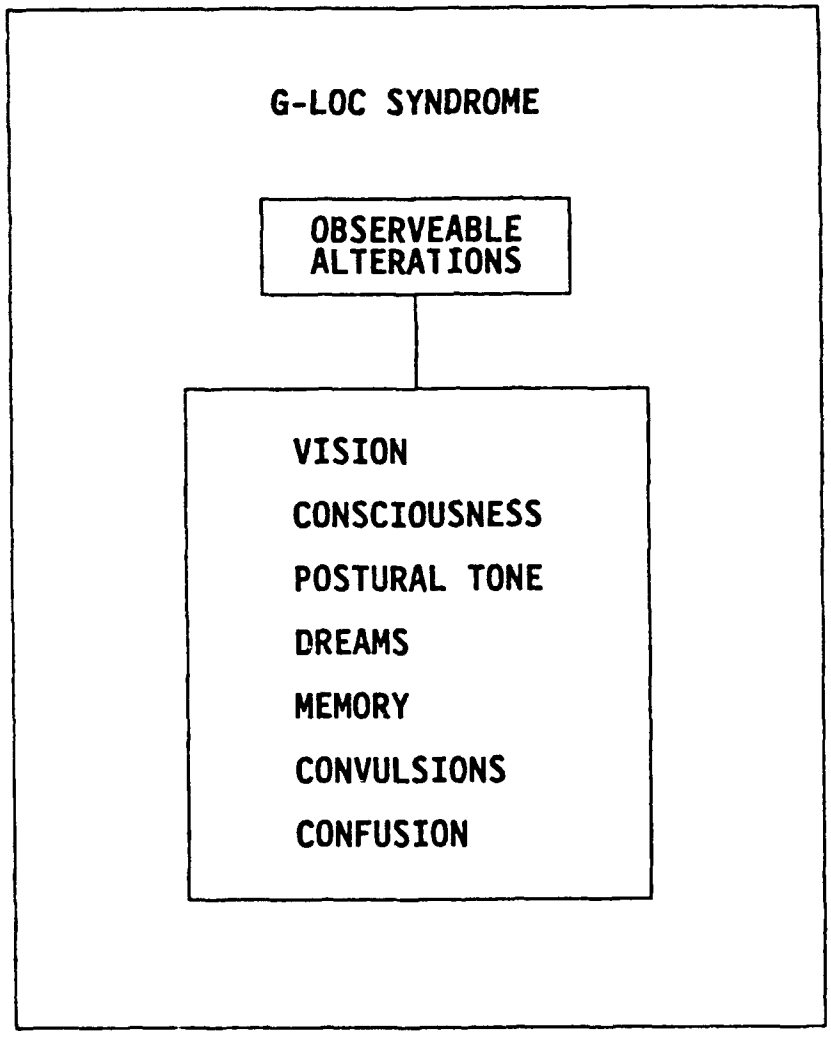
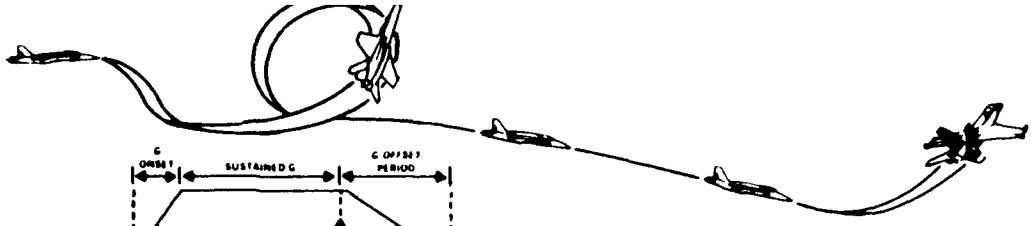
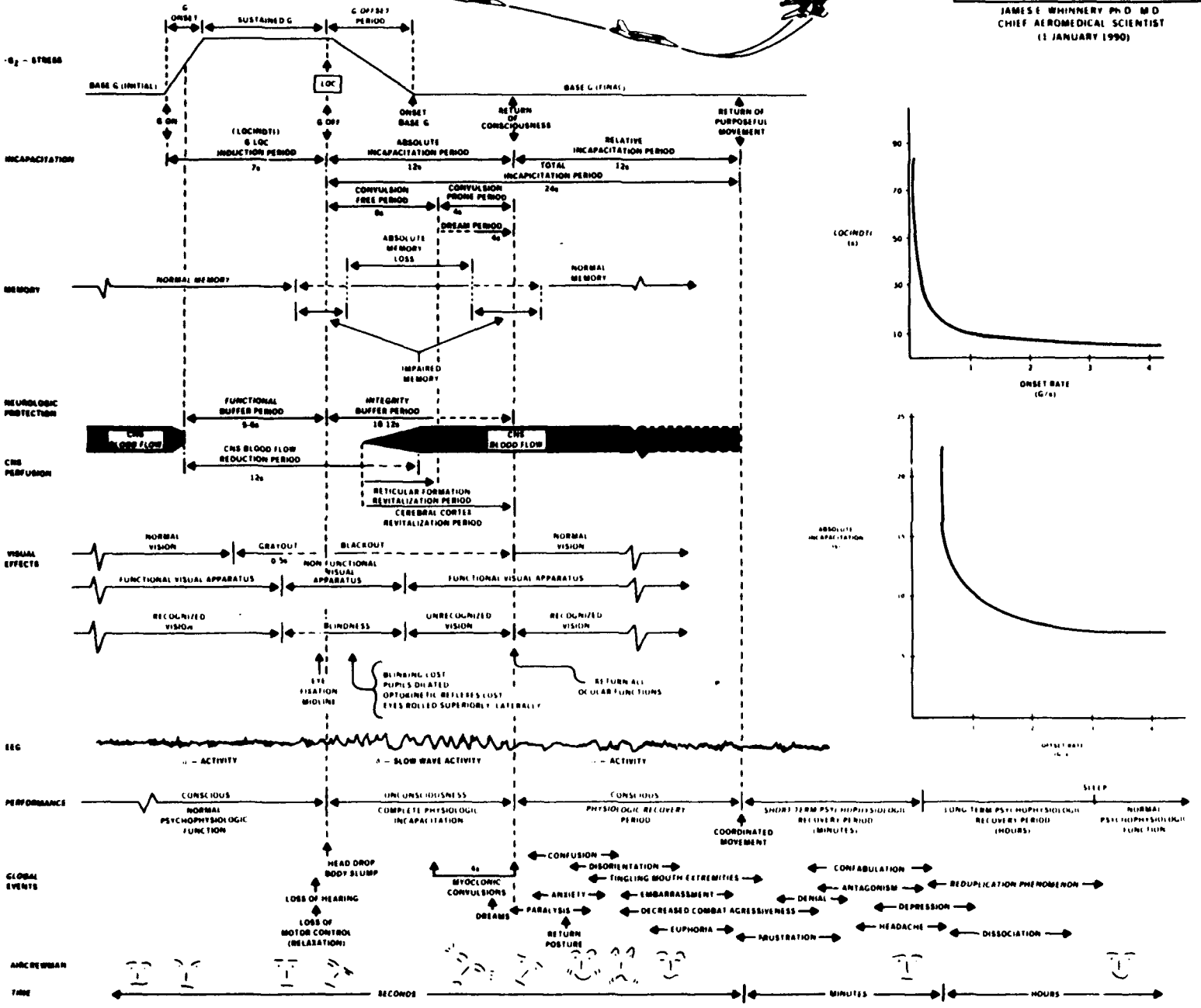


Figure 1. The G-LOC Syndrome Which Includes The Actual Alteration Of Or Loss Of Consciousness. (The Neurophysiological Alterations, Such As Electroencephalographic Changes, Are Not Listed Even Though An Integral Aspect Of The Overall G-LOC.)

ACCELERATION INDUCED LOSS OF CONSCIOUSNESS

JAMES E WHINNERY Ph D M D
CHIEF AEROMEDICAL SCIENTIST
(1 JANUARY 1990)





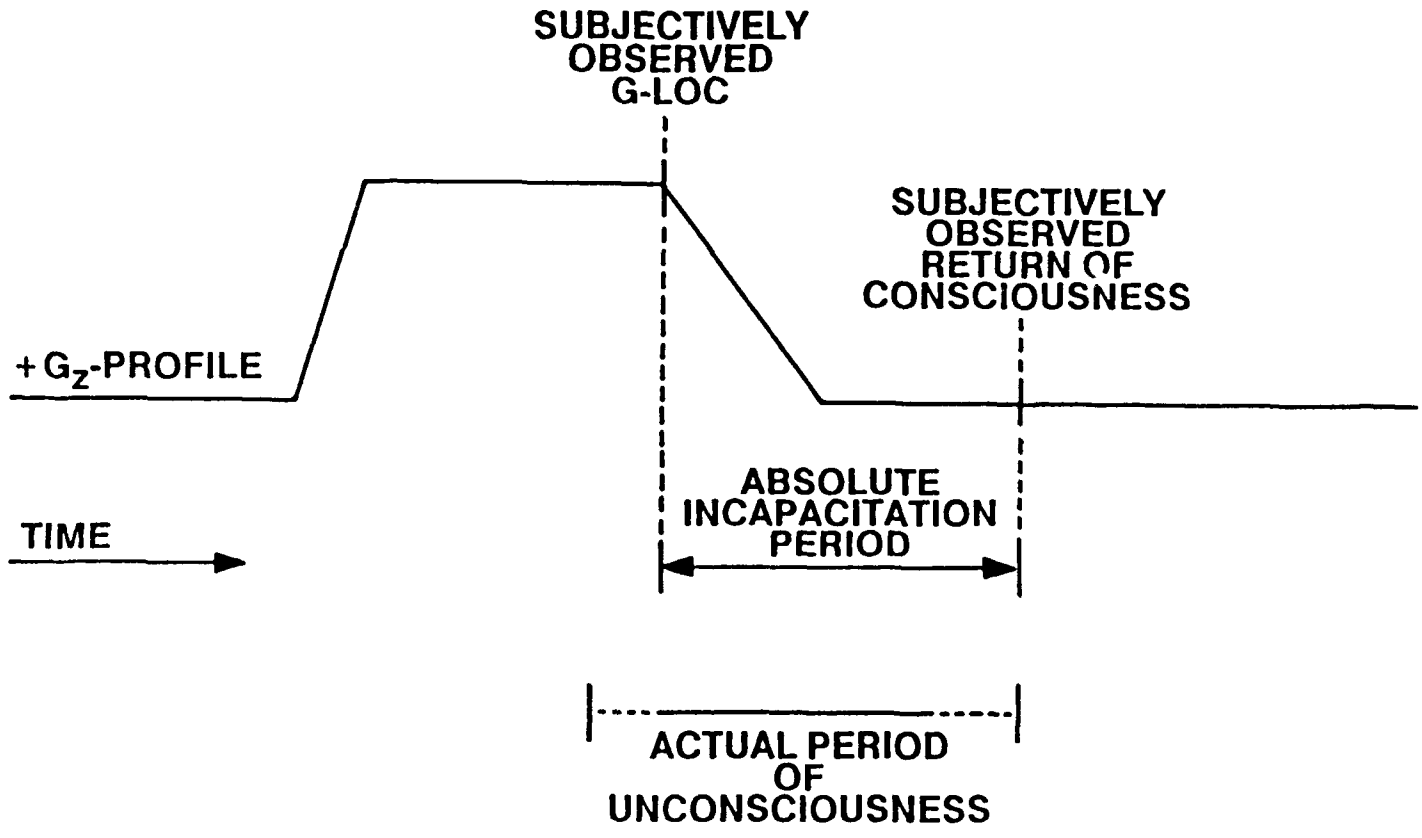


Figure 3. Illustration Of The Potential Error In Subjective Measurement Of Unconsciousness. The Accuracy Of Determining The Actual Period Of Unconsciousness Depends Upon The Definition Of Unconsciousness And The Ability To Measure The Phenomenon.

DISTRIBUTION LIST (Continued)
Report No. NADC-91042-60

No. of Copies

Dr. David Glaister, Royal Air Force 1
Institute of Aviation Medicine
Farnborough, Hants; United Kingdom

Mr. Michael Paul, Defense and Civil Institute 1
of Environmental Medicine
1133 Sheppard Avenue
P.O. Box 2000
Downsview, Ontario, Canada

Mr. Floyd McGowan 1
P.O. Box 35
Chapman, Alabama 36015

Air University Library 1
AUL/LSE, Maxwell AFB, AL 36112

USAFSAM 1
Brooks AFB, TX 78235
(Strughold Aeromedical Library/TSK-4)

Commander, USAF Armstrong Aerospace 1
Medical Research Lab
Wright-Patterson AFB, OH 45433

Commander, Naval Training Systems Command 1
12350 Research Parkway
Orlando, FL 32826

U.S. Air Force Tactical Air Command 1
Office of the Command Surgeon (SGP)
Langley AFB, VA 23665

Naval Air Development Center 32
Warminster, PA 18974-5000
(30 Copies for Office of Chief Aeromedical Scientist)
(2 Copies for Code 8131)

Commander, Naval Air Systems Command 8
Washington, DC 20361
(3 Copies for AIR-320R)
(2 Copies for AIR-931H)
(1 Copy for AIR-531B)
(2 Copies for AIR-5004)

1299th Physiological Training Flight 1
Malcolm Grow USAF Medical Center
Andrews AFB, Washington, DC 20331-5300

DISTRIBUTION LIST (Continued)
Report No. NADC-91042-60

No. of Copies

Air Force Office of Scientific Research 1
Bolling AFB, DC 20332-6448

Naval Aerospace Medical Institute 2
Naval Air Station, Pensacola, FL 32508

FAA Civil Aeromedical Institute 1
Oklahoma City, OK 73124

NASA Ames Research Center 1
Moffott Field, CA 94035

NASA 1
Johnson Space Center
Houston, TX 77058

Dr. Dov Jaron 1
Drexel University
Philadelphia, PA 19104

Dr. Steven Dubin, University of Pennsylvania 1
Philadelphia, PA 19104

Commandant of the Marine
Headquarters Marine Corps
Washington, DC 20380

Naval Weapons Center 1
China Lake, CA 93555-6001

Naval Air Force U.S. Pacific Fleet 1
NAS North Island San Diego, CA 92135

Commander Naval Air Force, U.S. Atlantic Fleet 1
Norfolk, VA 23511-5188

Center for Naval Analyses 1
4401 Fort Ave., P.O. Box 16268
Alexandria, VA 22302-0268

U.S. Air Forces In Europe (USAFE) 1
Office of the Command Surgeon/SG
APO, New York 09094-5001

Mr. Robert Montgomery 1
Aeromedical Training Institute
ETC Corporation
Southampton, PA 18966

DISTRIBUTION LIST
Report No. NADC-91042-60

	No. of Copies
Defense Technical Information Center Bldg. 5, Cameron Station, Alexandria, VA 22314	2
Chief of Naval Operations Dept of the Navy, Washington, DC 20350 (1 Copy for NOP-506N) (1 Copy for NOP-591) (1 Copy for NOP-05H) (1 Copy for NOP-09E)	4
Naval Aerospace Medicine Institute Naval Air Station, Pensacola, FL 32508	2
Office of Naval Research Code 1433, Washington, DC 20375 (1 Copy for Code 1433)	1
Naval Postgraduate School Monterey, CA 93940	1
USAF School of Aerospace Medicine USAFSAM/VNB, Brooks AFB, TX 78235 (1 Copy for Dr. Kent K. Gillingham)	1
USAF School of Aerospace Medicine Office of the Chief Scientist, Brooks AFB, TX 78235 (1 Copy for Dr. Russell R. Burton)	1
USAF School of Aerospace Medicine USAFSAM/VNB, Brooks AFB, TX 78235 (1 Copy for Dr. John W. Burns)	1
Air National Guard Support Center Mail Stop #18, Andrews AFB, DC 20331 (1 Copy for ANGSC/SG)	1
Advanced Physiologic Training Unit 833 Med Gp/SGT, Holloman AFB, NM 88330 (1 Copy for 833 Med Gp/SGT)	1
Federal Aviation Administration CAMI Library AAC 64D1, P.O. Box 25082, Oklahoma City, OK 73125 (1 Copy for Civil Aeromedical Institute)	1
Headquarters, U.S. Air Force Bolling AFB, DC 20332 (1 Copy for Office of the Surgeon General (SGP))	1